were sacrificed and the L4-L5 spinal cord was analyzed for the number of GABAergic cells in the superficial dorsal horn (Rexed laminae I–III). Data (mean±SEM) were compared to sham-operated animals (n = 3).
Results: The number of GABAergic cells in laminae I–III of chronic neuropathic rats was 48±4 (L4) or 50±3 (L5) as compared to 47±3 (L4) or 48±4 (L5) in sham-operated rats (p = 0.8 for both L4-L5 levels). In particular, mild allodynic rats were characterized by 54±6 (L4) or 53±7 (L5) GABAergic cells as compared to 44±6 (L4) or 48±4 (L5) GABAergic cells in moderate allodynic rats (L4: p = 0.3; L5: p = 0.6).
Conclusions: No changes in the number of GABAergic cells were observed in the superficial dorsal horn of chronic neuropathic rats as compared to sham-operated rats. Furthermore no relation between the number of GABAergic cells and the degree of mechanical allodynia in chronic neuropathic rats was noted.

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PERCUTANEOUS ELECTRIC NERVE STIMULATION AND BETA-ENDORPHIN LEVEL IN PATIENTS WITH NEUROPATHIC PAIN IN CHRONIC OSTEOARTHRITIS

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Introduction: Transcutaneous Electric Nerve Stimulation (TENS) used for a long time for the treatment of osteoarthritis as well as on peripheral neuropathy.
β-endorphin is a peptide, from the endorphins group, 31 amino acids long, resulting from processing of the precursor proopiomelanocortin (POMC), resulting from intracellular processing by internal enzymes known as prohormone convertases.
Objectives: Endorphins are endogenous opioid polypeptids. They are produced by the pituitary gland and the hypothalamus in vertebrates during strenuous exercise, excitement and pain. They resemble the opiates in their abilities to produce analgesia and a feeling of well-being.
Methods: Up to present time we don't have a sufficient source of objective evaluation of pain syndrome in osteoarthritic patiens. To clarify the intensity of pain syndrome and evaluate the treatment efficiency has been used enzyme-linked immunosorbent analysis (ELISA) of beta endorphin antibodies level in osteoarthritis patient's blood. Before and after of TENS treatment (45 days with exposure of 90 minutes) the beta-endorpin levels were investigated. Under our observation were 39 patients (both genders from 31 to 63 years old) with osteoarthritis and 43 healthy donors (men and women from 24 to 61 years old).
Results: It was revealed that in 26 (63%) patients and in 12 donors (28%) the beta endorphin level was incteased after the course of treatment.
Conclusions: The ELISA of blood samples for beta-endorphin measure in patients with neuropathic pain in osteoarthritis could serve as additional source of information for pain evaluation alongside with traditional pain scales and provide more reliable laboratory information for this matter.

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OUTCOME OF VITAMIN C SUPPLEMENTATION ON LEAD-INDUCED APOPTOSIS IN ADULT RAT HIPPOCAMPUS

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ConcluLead (Pb), due to its numerous industrial applications has caused widespread pollution of the environment. One principle target for lead in the human body is the central nervous system (CNS). Research has shown however that the toxic effects of lead can be moderated by antioxidant agents such as vitamin C.
The aim of this study was to investigate the protective effects of vitamin C supplementation against lead induced apoptosis in the adult male rat hippocampus. 30 male rats were divided into three groups: One group was given lead acetate (20 mg/kg) another lead acetate plus vitamin C (500 mg/daily) for seven days and the third group was used as control. Co-treatment with vitamin C for 7 days caused a significant decrease in Bax expression with a decrease in blood lead level as compared with those treated with lead alone. The results suggest that supplementing essential nutrient vitamin C may possibly prevent lead-induced apoptosis.

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MECHANISMS OF PHASE-DEPENDENT PAIN-RELIEF ACTIVITY OF GLYCINE TRANSPORTER INHIBITORS AFTER NERVE INJURY

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Our previous studies demonstrated that enhancement of spinal glycinergic system by administration of inhibitors of glycine transporter 1 (GlyT1) and GlyT2, or knockdown of spinal GlyTs by siRNA of GlyTs mRNA produced a profound anti-allodynia effect after 4 days post partial sciatic nerve ligation in mice, while these treatments were without effect during 3 days post surgery. To assess the implications of alteration of glycine signaling to this phase-dependent anti-allodynia effect by glycinergic system during development of neuropathic pain, the changes in glycine signal-related protein levels in the spinal cord after nerve injury were studied in mice. No significant alteration in the expression of glycine receptor α3 and GlyT1/2 were observed. The expression of K^+, Cl^--cotransporter type 2 (KCC2) protein decreased during the 0.5–3 days and recovered after 4 days post-surgery. This down-regulation of KCC2 was prevented by the administration with minocycline or knockdown of spinal brain-derived neurotrophic factor (BDNF) and TrkB by siRNA. The administration with minocycline or knockdown of BDNF and TrkB abrogates the development of neuropathic pain until 3 days after the operation.
This study suggests that nerve ligation via activation of TrkB in the neuron by BDNF release from microglia causes the down-regulation of KCC2 lead to a negative shift in Cl^- reversal potential, resulting in a switch of glycine action from inhibitory to excitatory at the early stage after nerve injury. This may a cause for the reversal glycinergic influence on pain state after nerve injury.

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HIV-1 GP120-MEDIATED EFFECTS ON NEURITE OUTGROWTH

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HIV-associated distal sensory polyneuropathy (HIV-DSP) is a frequent neurological complication of HIV infection and is characterised by a 'die back' pattern of axonal degeneration of predominantly small diameter peripheral neurons. Recent research has identified the HIV viral envelope glycoprotein, gp120, as a key mediator of axonal degeneration and neuronal apoptosis both *in vitro* and *in vivo*. However the exact mechanisms of gp120 action remain unclear.
To investigate gp120-mediated neurotoxicity primary Dorsal Root Ganglion (DRG) neurons, in Campenot chambers and non-compartmentalised culture systems, were treated with either vehicle or $gp120_{MN}$ (20pM-2nM). Neurite density and gp120 localisation were assessed at different time points. Neurites were detected with BIII-tubulin staining and neurite density measured with HCA vision software. Neurite density was defined as total neurite length divided to the number of neurons.